



Armed Forces College of Medicine

AFCM



Pathology of Chronic Obstructive Pulmonary Diseases (COPD-2)

by

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good
morning

INTENDED LEARNING OBJECTIVES (ILO)



By the end of this lecture the student will be able to:

1. Define emphysema.
2. Explain the pathogenesis of emphysema.
3. Describe the pathological features of emphysema.
4. Discuss the types of emphysema.
5. Determine the effects & complications of emphysema.

Lecture Plan



- 1. Part 1 (5 min) Introduction**
- 2. Part 2 (35 min) Main lecture:**
- 3. Part 3 (5 min) Summary**
- 4. Lecture Quiz (5 min)**

Terminal respiratory unit (*Acini*)

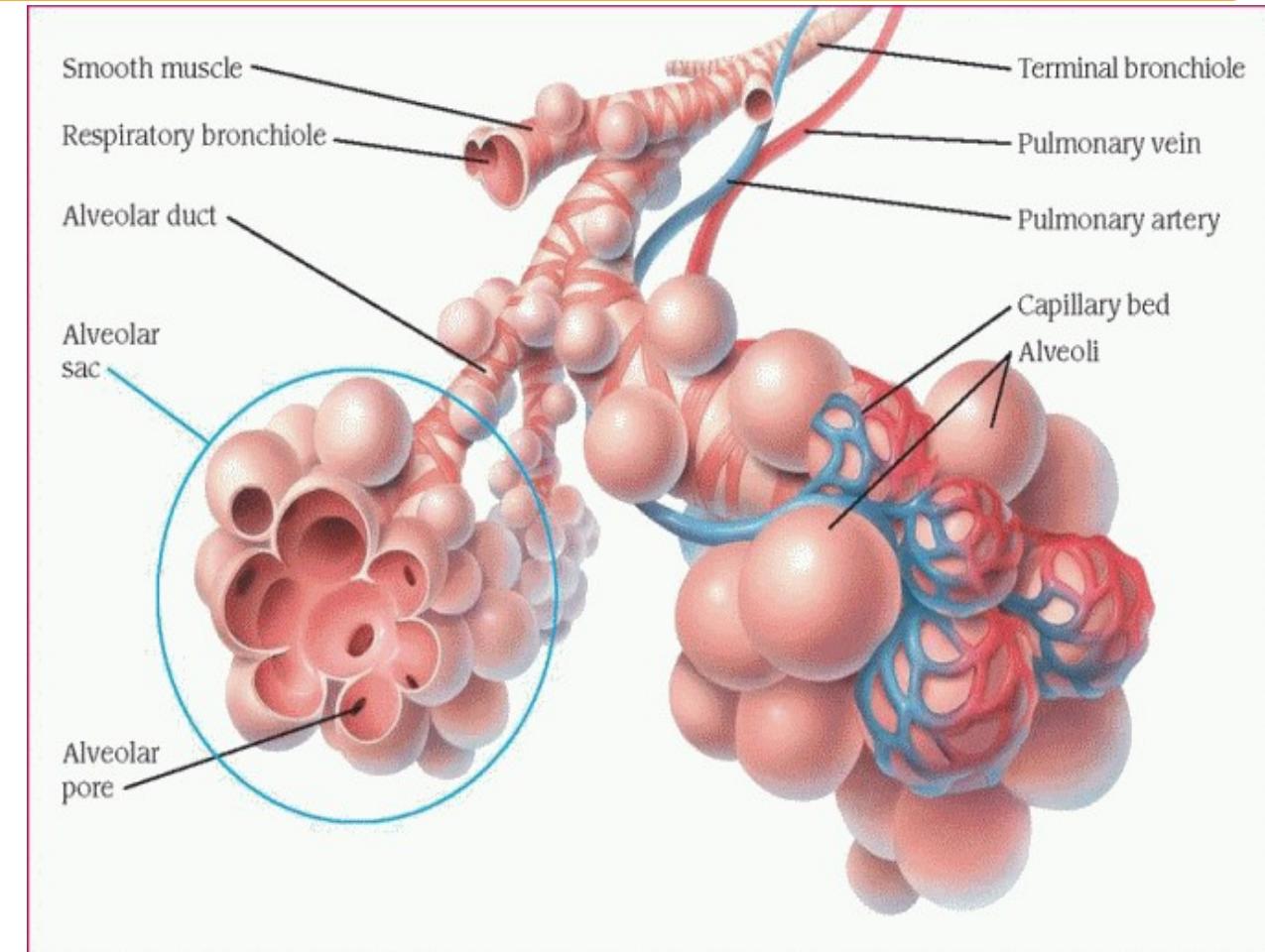


Terminal respiratory

unit (*Acini*) is the functional **unit** of the lung. It includes:

- a- terminal respiratory bronchiole
- b- alveolar ducts
- c- alveolar sacs

It is also called **primary lobule**



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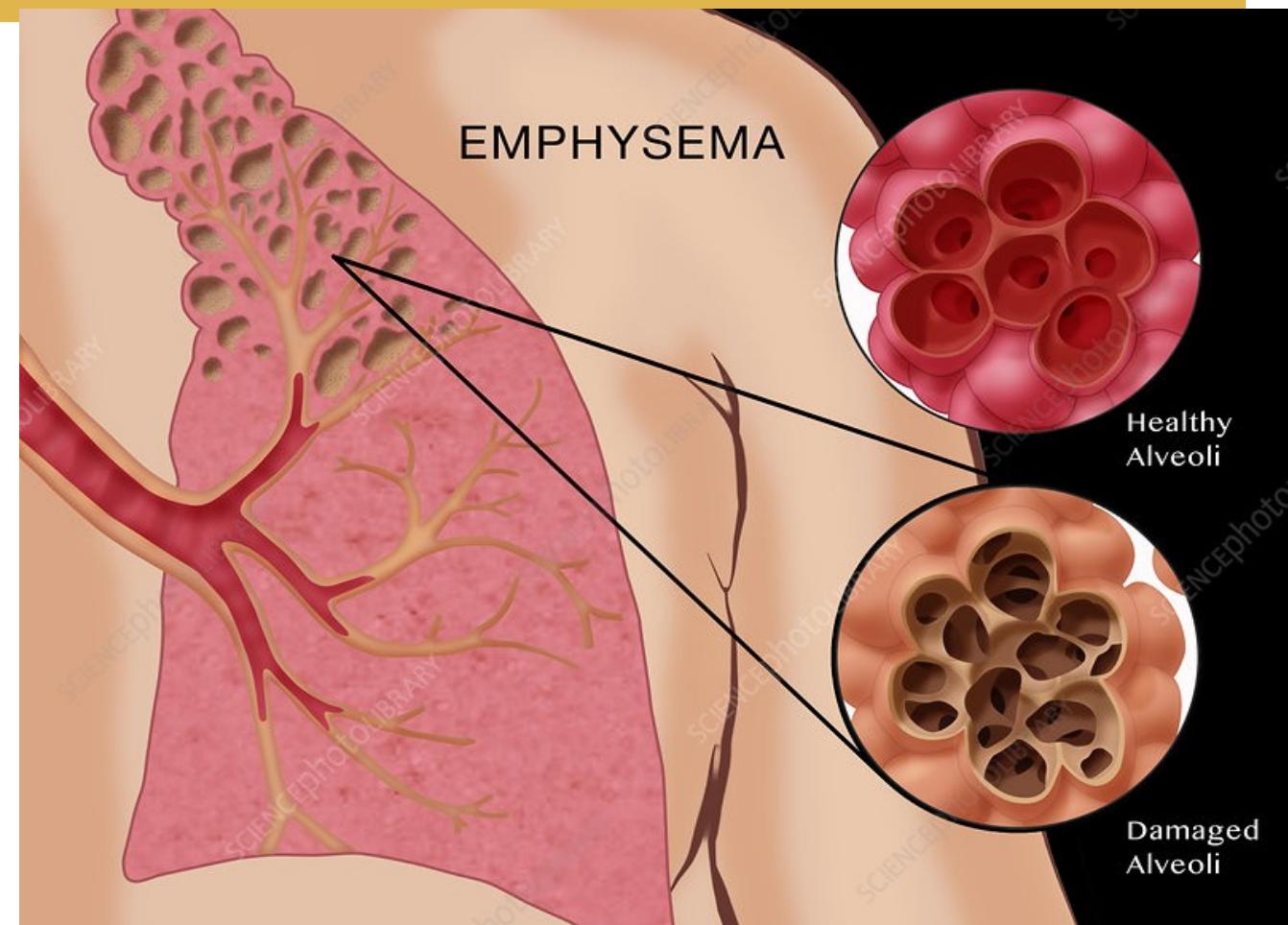
Definition:

Emphysema

Permanent over-distention of

the air spaces distal to the terminal bronchioles with destruction of the alveolar septa without significant fibrosis. The condition ends by ***loosing the elastic recoil of the lung***

Incidence: it is more common in males above 40 years



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Emphysema



Pathogenesis:

1- Protease anti protease theory:

- Lung is rich in elastic tissue helping in elastic recoil.
- Exposure to tobacco smoke stimulates inflammation with infiltration by neutrophils which secrete a protease called **Elastase Enzyme**.
- Elastase is derived also from macrophages ,mast cells and bacteria in the lung .

Emphysema



- Elastase causes:
 1. ***Injury*** of epithelial cells
 2. ***Proteolysis*** of extracellular matrix
 3. ***Destruction*** of the interalveolar ***α -1 antitrypsin***.

Unless antagonized by normal antielastase the

- So, more than 80% of patients with congenital deficiency of α -1

antitrypsin develop

Emphysema



2-Cigarette smoking:

Have a role in the genesis of **Centriacinar emphysema.**

Because:

- a- Smokers have greater number of neutrophils and macrophages in their alveoli.
- b- Smoking stimulates release of elastase from neutrophils.
- c- Smoking enhances elastase activity in macrophages.
- d- Oxidants in cigarette smoke inhibit α -1 antitrypsin.
- e- Cigarette smoking causes chronic bronchitis.

Emphysema



3-Chronic bronchitis:

Chronic bronchitis is a helping factor due to:

- Swelling of the inflamed bronchial mucosa leading to bronchial obstruction.
- Exaggerated obstruction during expiration leading to trapping of much air.

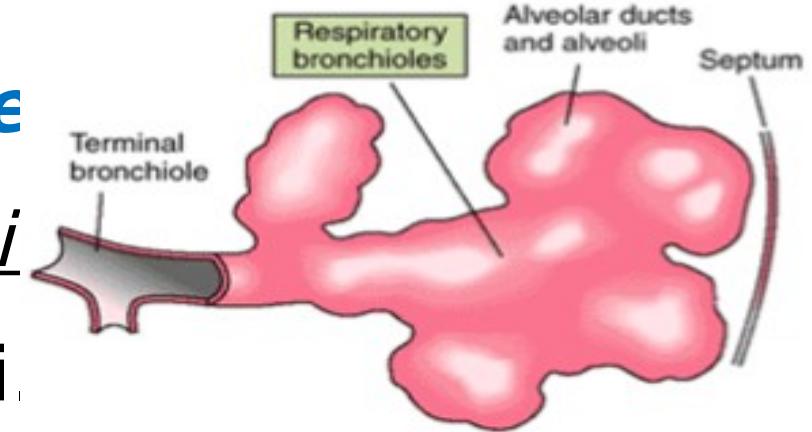
Emphysema



Types:

1- *Panacinar (panlobular) emphysema*

- Involves entire respiratory acinus (unified) from respiratory bronchiole to alveoli.
- Occurs more commonly in the **lower lobes**, especially basal segments, and anterior margins of the lungs.
- The affected acini are uniformly enlarged.
- It is the type of emphysema that occurs due to $\alpha-1$



PANACINAR EMPHYSEMA

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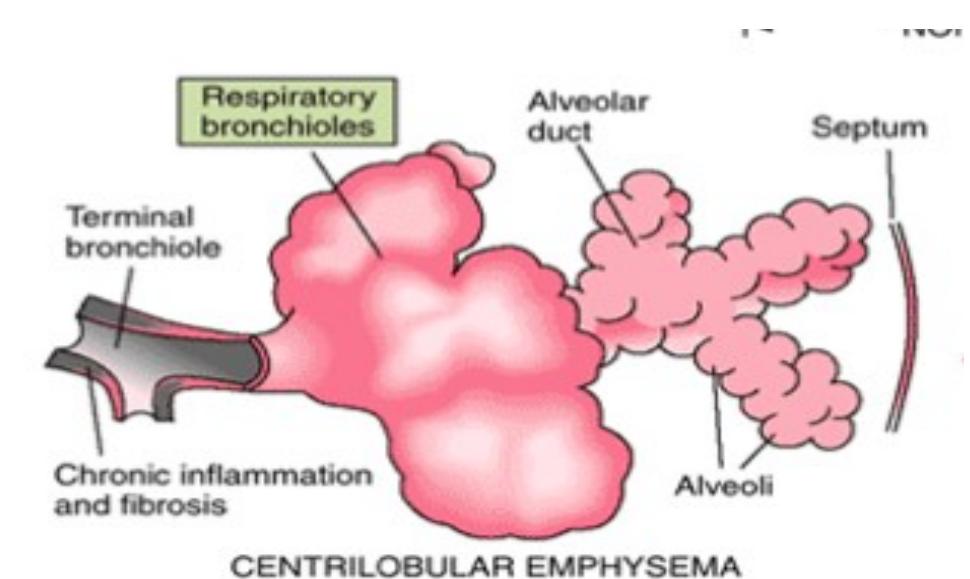
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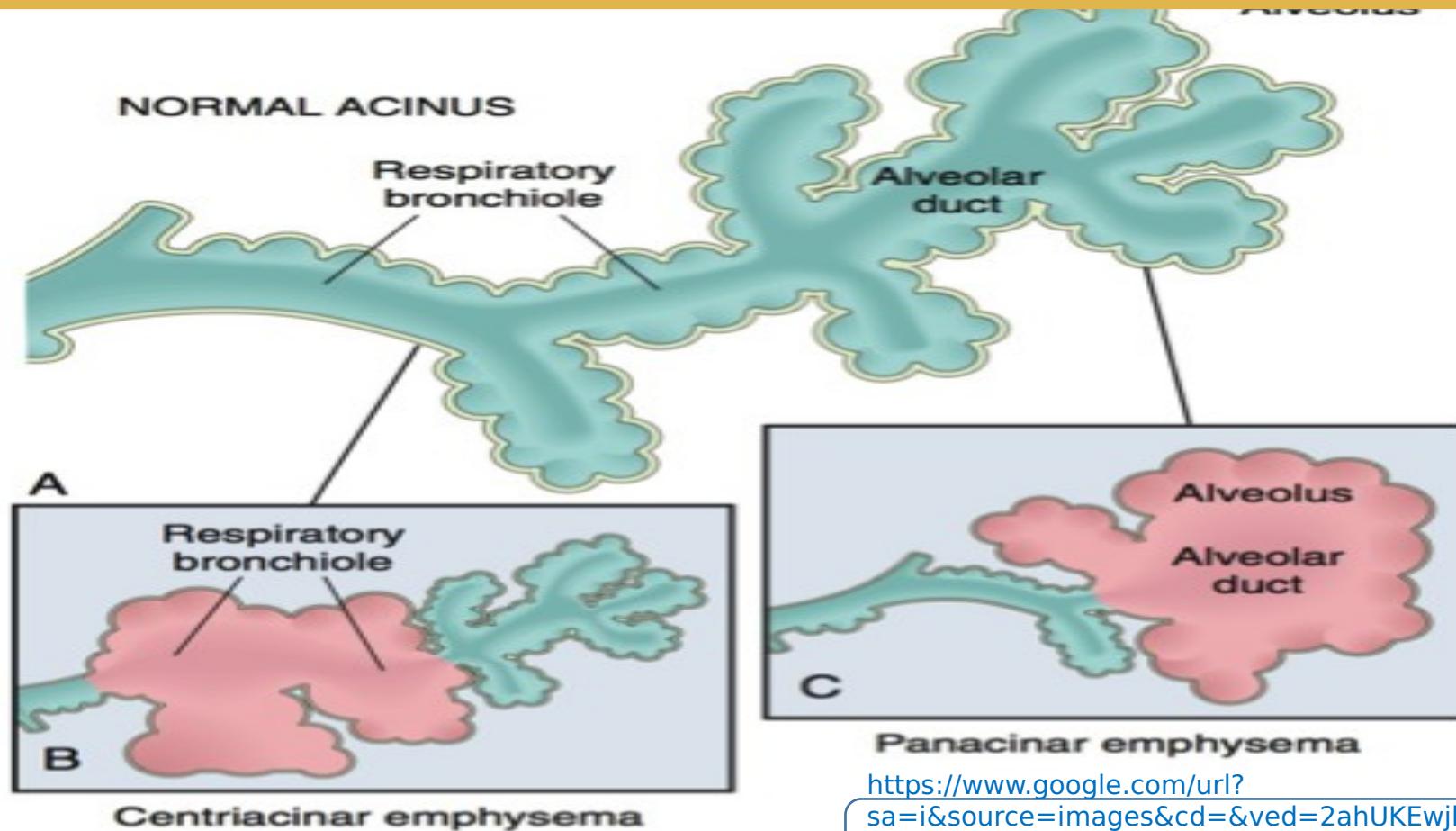
2- *Centrilobular (centriacinar) emphysema :*

- It affects the central part of the acini, while the distal alveoli are spared. It is more common in the **upper lobes**.
- This type of emphysema is the relation to **cigarette smoking** and in patient without congenital deficiency in antitrypsin.

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Emphysema



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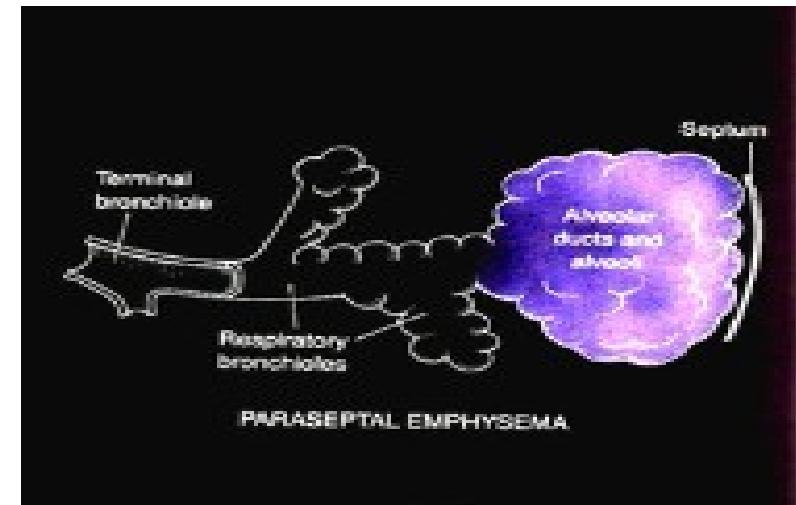
3- Paraseptal emphysema:

The proximal portion of the acinus is normal but the **distal part** is over-distended . **Bullae** formation is common.

It is a common cause of
spontaneous pneumothorax

in young adults.

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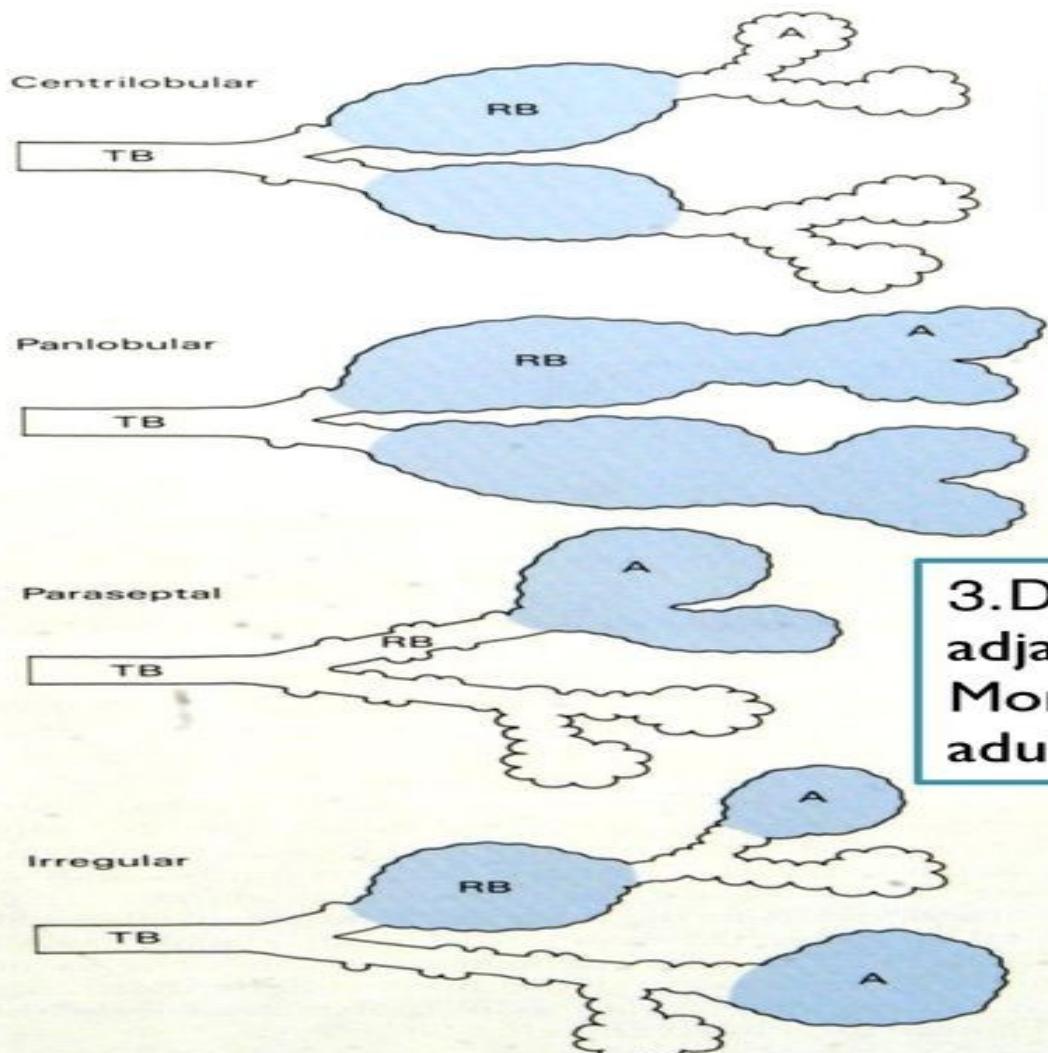


Emphysema



4- *Irregular emphysema:*

The acinus is irregularly over-distended .The affected acini are usually adjacent to areas of lung scars(healed inflammatory lesions). It doesn't affect the pulmonary functions



1. Centriacinar:
in heavy smoker, severe in upper lobes

2. Alpha 1 antitrypsin deficiency:
Commonly affect both lower lobes at lower zones

3. Distal acinar /paraseptal:
adjacent to areas of fibrosis or atelectasis
More severe in the upper half of the lungs, young adult, unknown cause, spontaneous pneumothorax

4. Irregular
Associated with scarring, e.g. in TB
Asymptomatic

Classification of emphysema

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Emphysema

Grossly:

- Both lungs are voluminous, fill the chest cavity and cover the heart.
- **Barrel-shaped chest** in which the anteroposterior and the transverse diameters of the chest *are equal*.
- The lung is dry, pale and light and there is indentation on their surface at the site of ribs.
- There is bullae formation along anterior border, base and apex of lung.

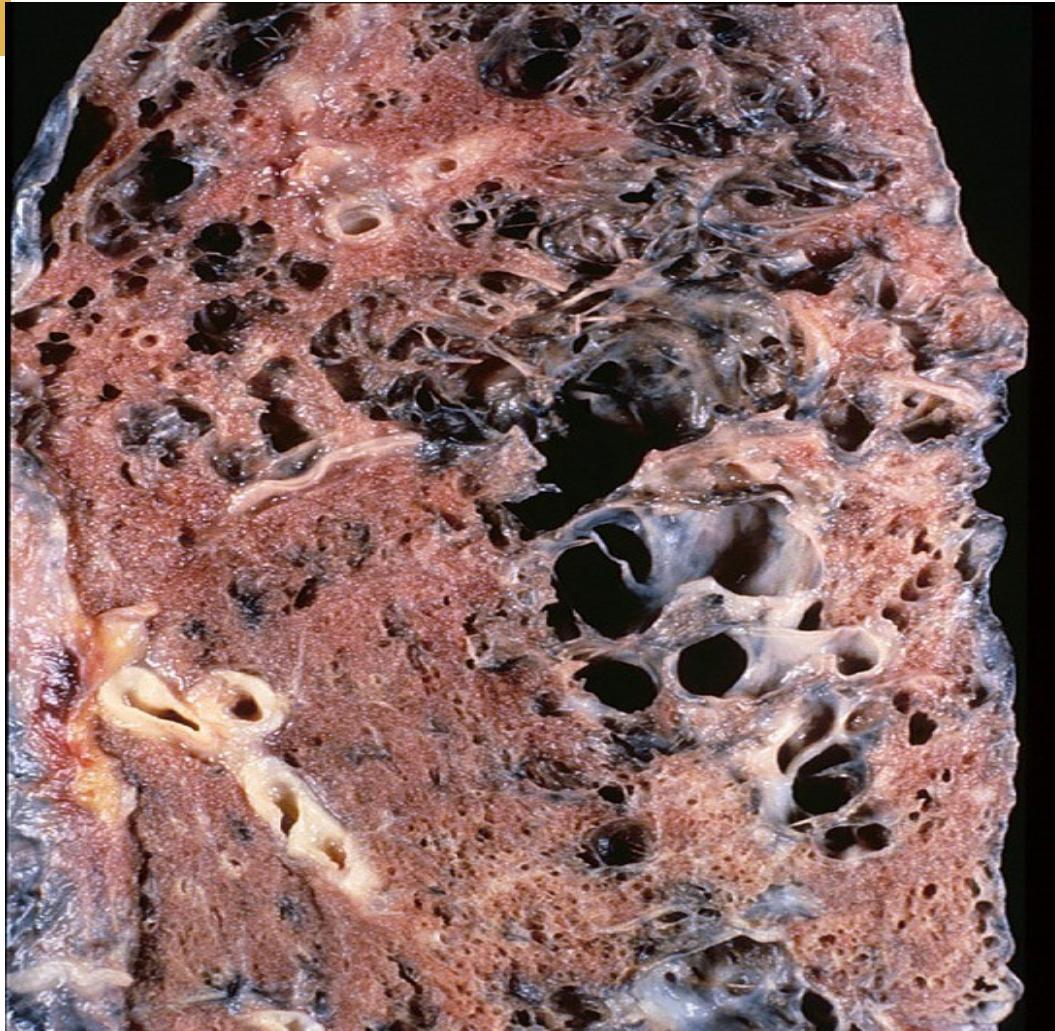


Bullae Emphysema

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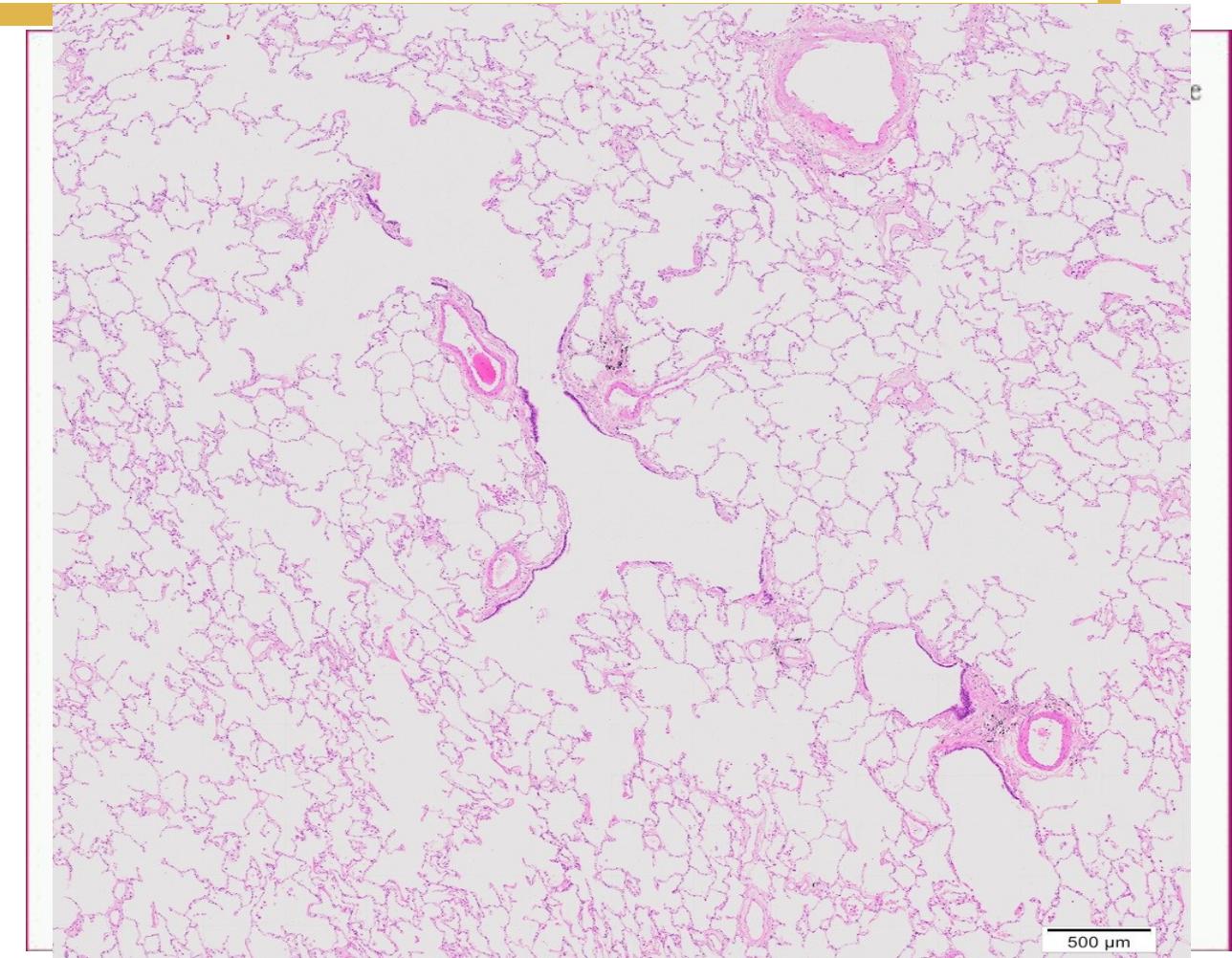
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Emphysema

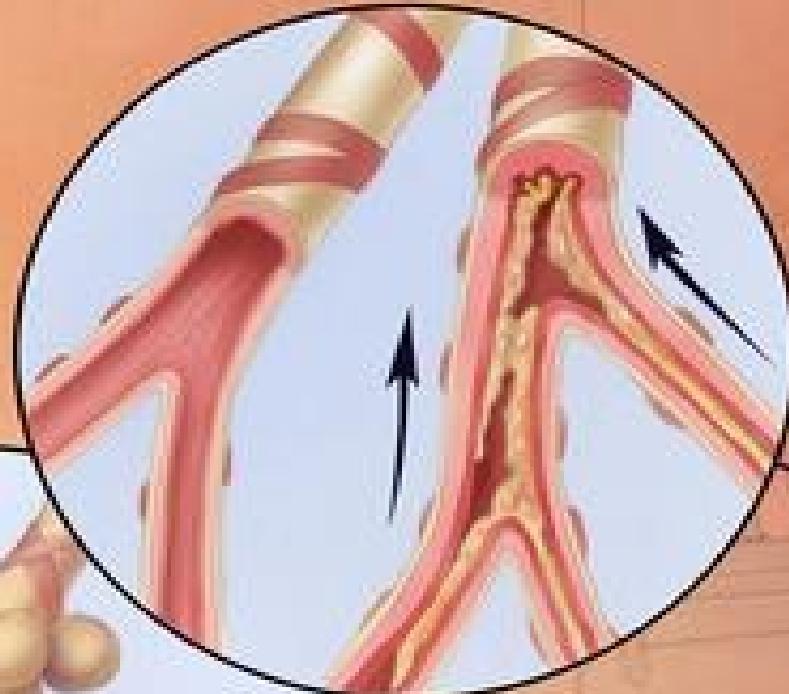
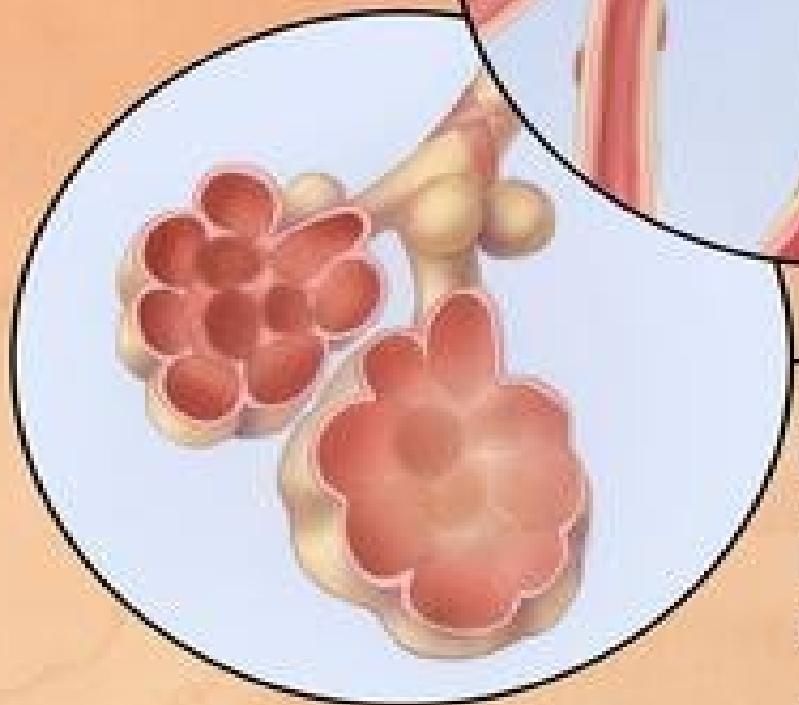
- **Microscopically:**
- Distention affects the respiratory bronchioles only in cases of centrilobular emphysema and the respiratory bronchioles, as well as alveolar ducts and alveoli in cases of panacinar emphysema.
- The bronchi show evidence of ***chronic inflammation*** with thickening in the wall of arterioles due to pulmonary hypertension.



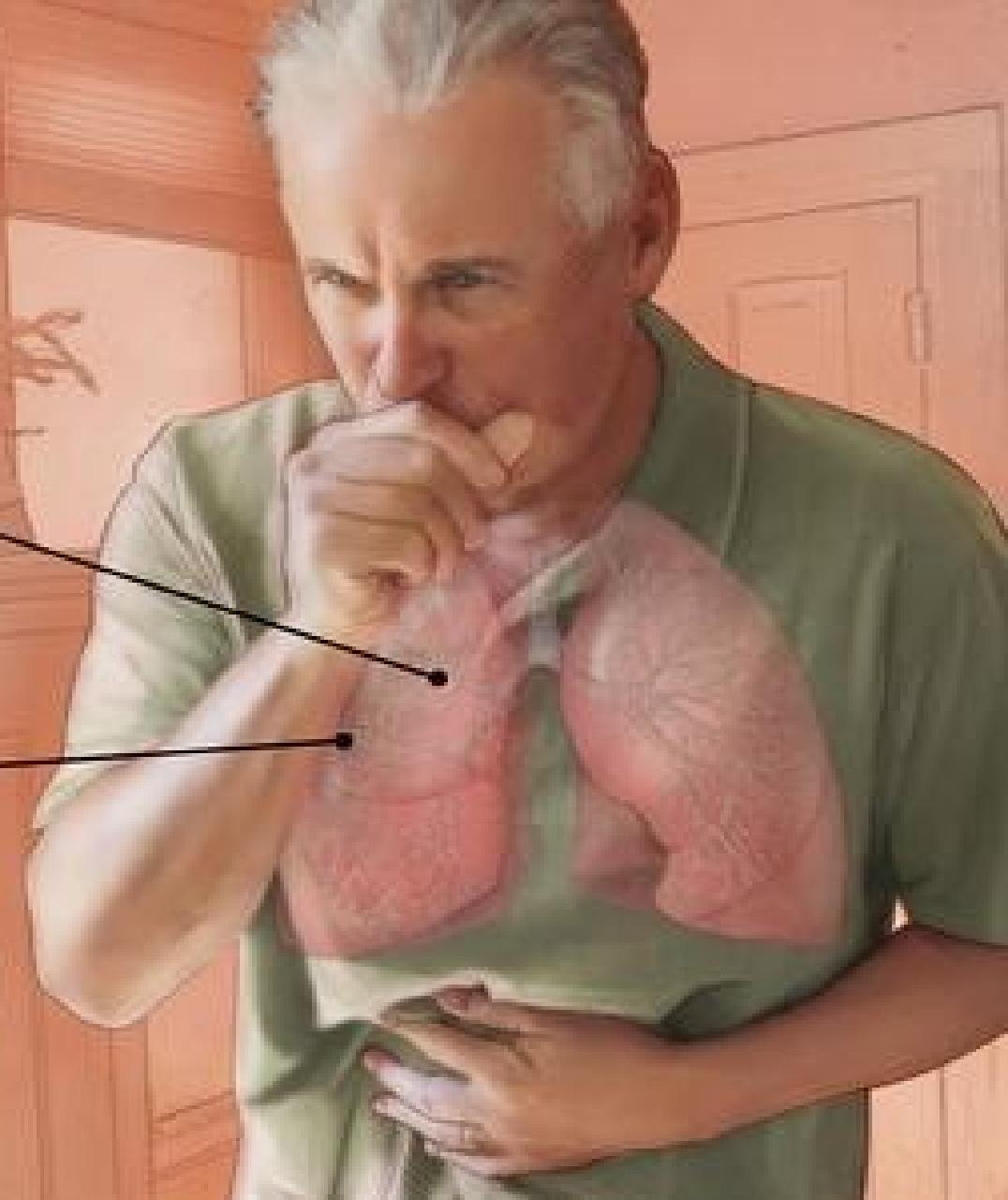
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Bronchitis

Increased mucus and inflammation



Emphysema
Destruction and enlargement of air spaces



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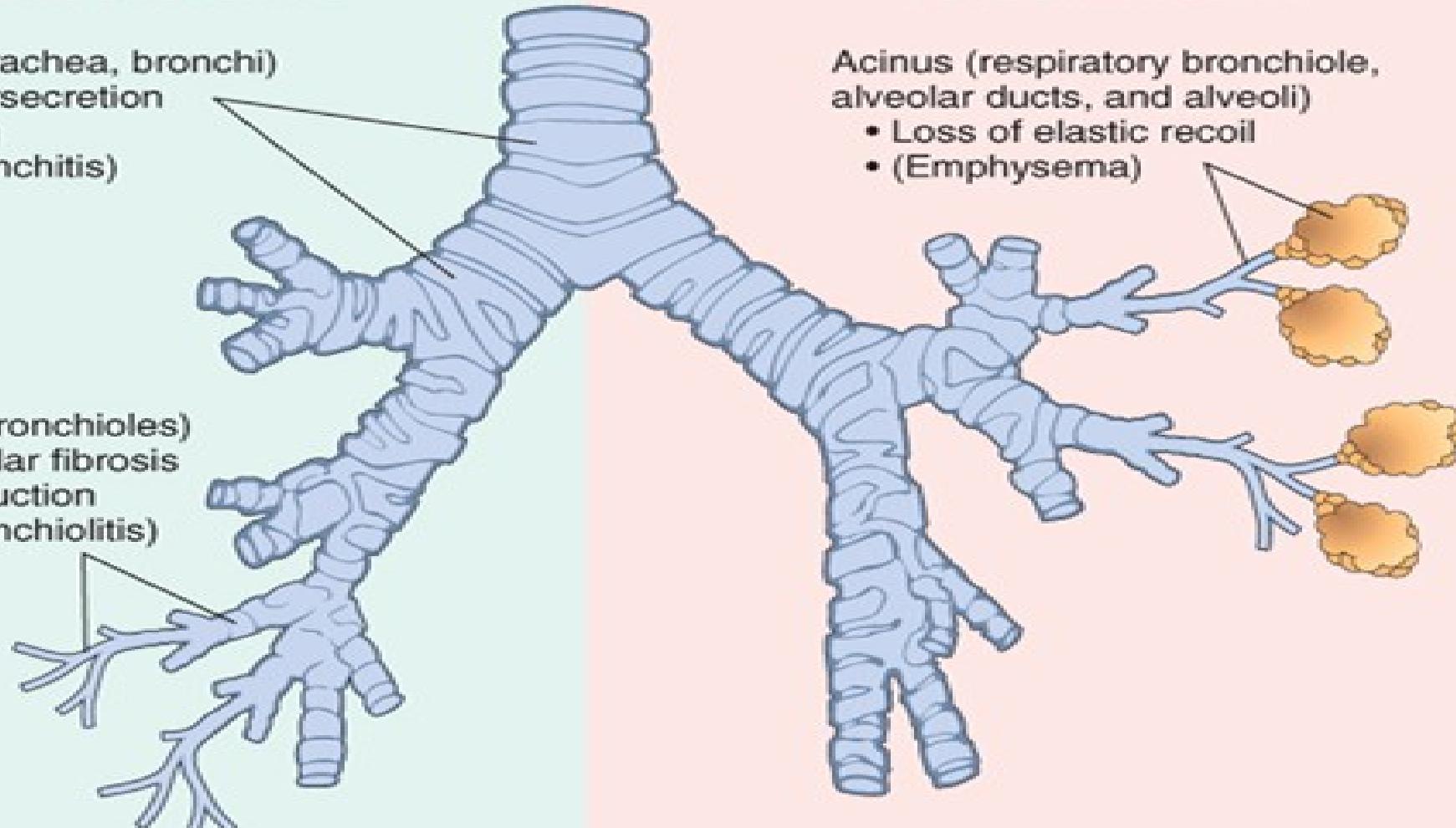
Emphysema

Large airways (trachea, bronchi)

- Mucus hypersecretion
- Inflammation
- (Chronic bronchitis)

Small airways (bronchioles)

- Peribronchiolar fibrosis
- Airway obstruction
- (Chronic bronchiolitis)



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Other types of Emphysema

3- *Interstitial*

(mediastinal or surgical) emphysema:

Means air in the interstitial tissue *[i.e. not within the alveoli]*.

It occurs due to over distension of alveoli in severe cough or penetrating chest injury, which lead to alveolar rupture with air escapes to the interstitial tissue of lung, mediastinum or the subcutaneous tissue of the neck.

1- Atrophic or senile emphysema:

■ It occurs in very *old patients and in debilitated persons* suffering from wasting diseases due to senile atrophy of the alveolar walls and decrease of elastic tissue.

2- Compensatory emphysema [hyperinflation]:

Multiple foci of emphysema develop in order to compensate near-by areas of *lung collapse, fibrosis and consolidation.*

It is not true emphysema since it is not accompanied by destruction of the wall of the alveoli.

Emphysema



Complications:

- Dyspnea due to increased air residue.
- Spontaneous pneumothorax or interstitial emphysema from rupture of emphysematous bullae.
- Right-sided heart failure ***[cor pulmonale]*** due to pulmonary hypertension.
- Respiratory failure.

Lecture Quiz



1- Congenital deficiency of α -1 antitrypsin results in:

- a- Irregular emphysema
- b- Centrilobular emphysema
- c- Panacinar emphysema**
- d- Paraseptal emphysema
- e- Compensatory emphysema

Lecture Quiz



It is not true emphysema since it is not accompanied by destruction of the wall of the alveoli:

- a- Atrophic emphysema
- b- Compensatory emphysema
- c- Paraseptal emphysema
- d- Interstitial emphysema
- e- Panacinar emphysema

SUGGESTED TEXTBOOKS



- 1- Kaplan Medical step 1, lecture notes in Pathology: Chapter 14, Respiratory system , pp. 125-143, 2017.
- 2- Hursh Mohan Text Book of Pathology, 7th ed. (2015): Chapter 14, Respiratory system, pp. 442-488.
- 3- Hursh Mohan Text Book of Pathology, 7th ed. (2015): Chapter 15, eye, ENT and neck, pp. 495-500
- 4- Robbins basic of Pathology, 10th ed. (2018): Chapter 13, Lung. pp. 495-549 ,



A yellow sticky note with a blue pushpin at the top left. The note contains the text "THANK YOU :)" in purple, handwritten-style font. A purple line underlines the word "YOU".

THANK
YOU :)